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COMMENTARY

Understanding and Transcending "p" Requires a Functional Model of Psychopathology: Commentary on Caspi et al. (2026)

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Caspi et al. (2026) masterfully document the extensive overlap between mental disorders, summarized at the broadest level by the general construct of "p." This overlap seems to leave little room for specificity; moreover, mainstream bottom-up approaches are ill-equipped to address the dual challenge of heterogeneity within diagnostic categories coupled with similarity across the same categories. Here I argue that we can make progress by taking an explicitly functional perspective, and remapping psychopathology onto broad, evolutionarily relevant dimensions of individual variation. I briefly present the fast–slow–defense (FSD) model, a taxonomy of psychopathology based on the evolutionary concept of life history strategies. The FSD model identifies meaningful subtypes within diagnostic categories and reassembles them into broad, functionally distinct spectra with different risk factors and developmental patterns ("lumping via splitting"). In doing so, it simultaneously explains, deconstructs, and goes beyond the descriptive notion of "p," to provide an explanatory framework rooted in biological function.

General Scientific Summary
To overcome current limitations, models of psychopathology must meet the dual challenge of heterogeneity within diagnostic categories coupled with similarity and overlap across the same categories. This comment argues that bottom-up approaches are insufficient; to make progress, we need to take an explicitly functional perspective, as illustrated by the fast–slow–defense model.

Keywords: evolutionary psychopathology, life history theory, "p" factor, transdiagnostic models

Caspi et al. (2026) have written a masterful overview of how mental disorders overlap within people and families, both throughout individual lives and across generations. The construct of "p," originated in factor-analytic models of psychopathology, crystallizes the notion that "all disorders go together" and hence should be studied and understood together.

I absolutely agree with the spirit of this approach, but a crucial question remains: Is there a level of description or a mode of classification that allows one to find true specificity beyond the generalized overlap summarized by "p"? For reasons touched upon in the article, neither purely empirical diagnostic subtypes (derived bottom-up from biomarkers, symptom profiles, etc.) nor the transdiagnostic spectra of the HiTOP (derived bottom-up from correlations among symptoms or diagnoses) seem able to convincingly answer this question (for critical assessments see, e.g., Del Giudice, 2018; Del Giudice & Haltigan, 2023; Haeffel, Jeronimus, Fisher, et al., 2022; Haeffel, Jeronimus, Kaiser, et al., 2022).

In this commentary, I argue that we can make progress by taking an explicitly functional, top-down perspective on the structure of mental disorders. The idea is to remap psychopathology onto broad, evolutionarily relevant dimensions of individual variation that determine individual patterns of risk for mental disorders, both concurrently and across developmental stages. In previous publications (Del Giudice, 2018; for a quick overview see Del Giudice & Haltigan, 2023), I proposed the fast–slow–defense (FSD) model, which is based on the concept of a fast–slow continuum in life history-related traits. The "core" psychological traits associated with fast and slow life history strategies are impulsivity, present versus future orientation, risk-taking, and sensation seeking; precocious versus delayed onset of sexual desire and sexual debut; restricted versus unrestricted sociosexuality (i.e., the preference for uncommitted sex with multiple partners); sensitivity to sexual/moral disgust; orientation toward long-term mating; stable versus unstable romantic attachments; and exploitative versus cooperative social attitudes. Taken together, these traits paint a contrast between "risky, short term" and "safe, long term" psychologies, both in the domains of mating and social relationships and in the weighting of potential dangers against rewards (see Del Giudice, 2020, 2025).

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The FSD model combines the fast–slow continuum with a dimension of defense activation that captures individual differences in the readiness and intensity of protective psychological mechanisms such as anxiety, fear, panic, and sadness/depression. Heightened defense activation can occur at both the slow and (especially) fast ends of the fast-slow continuum. In total, the model identifies three functional spectra of psychopathology, which simultaneously describe "kinds of disorders" and "kinds of people": a fast spectrum and slow spectrum that exist in a diametrical reciprocal relation, plus a defense activation spectrum that overlaps with the other two. Individual differences in cognitive ability are also taken into account; low and/or impaired cognitive abilities contribute to the associations between multiple disorders, even if they do not define a distinct pathology spectrum. Of course, different taxonomies can be useful for different purposes; the FSD fully recognizes that this is not the only way to draw meaningful functional distinctions that are relevant to psychopathology (see Del Giudice, 2018).

My goal here is not to describe the specifics and theoretical underpinnings of the FSD model in any detail. Instead, I want to focus on how the model meets what Caspi et al. (2026) aptly describe as the "dual challenge" of heterogeneity within diagnostic categories coupled with similarity and overlap across those same categories. It does so in a unique way, which makes it radically different from alternatives like the HiTOP: Namely, it employs trait constellations related to fast strategies, slow strategies, and defense activation to (a) identify putative functional subtypes within diagnostic categories, and (b) assign those subtypes to the three corresponding spectra. This approach might be called lumping via splitting, because diagnostic categories are subtyped only to be re-assembled into a small number of broad-band spectra—thus reducing the dual challenge to a single, tractable problem.

To give just two examples: eating disorders (EDs) can be split into an impulsive, fast spectrum subtype (F-EDs) that overlaps with cluster B personality disorders; and an overcontrolled, slow spectrum subtype (S-EDs) that overlaps with autistic traits and cluster C personality disorders. While bulimic presentations are relatively more common in fast spectrum EDs, these subtypes are defined by their personality/motivational correlates, rather than by (unstable, nonspecific) symptom profiles. Obsessive–compulsive disorder (OCD) can be similarly split into a defense activation subtype (D-OCD) that is more prevalent in females, centers around themes of harm prevention, and overlaps with other defense activation disorders as well as psychosis; and a male-biased slow spectrum subtype (S-OCD) that is characterized by perfectionism, themes of symmetry/ordering and "just not right experiences," and overlaps with autism and obsessive–compulsive personality disorder. Germane to the argument developed in Caspi et al.'s (2026) article, conditions in different spectra of the FSD model are also expected to show distinctive developmental patterns, from typical age of onset to remission trajectories (details in Del Giudice, 2018).

It is interesting to consider the association between EDs and OCD in this light. S-OCD is expected to overlap with S-EDs (which are part of the same functional spectrum), but not with F-EDs; however, D-OCD can be associated with both ED subtypes. If one were to ignore the functional subtypes and treat EDs and OCD as unitary categories, they would show moderate overlap, both with one another and with virtually all the other mental disorders (since the association networks of their subtypes would merge into a mixed, heterogeneous network). Intriguingly, if one considers the data on assortative mating and parent–offspring associations presented by Caspi et al. (2026, Figures 1 and 3), OCD and EDs stand out for their comparatively small associations with other disorders—consistent with the idea that they comprise functionally distinct subtypes with partly different networks of overlap.

The broader and more important point is that the FSD model should not be tested naïvely; despite the diametric nature of the fast–slow continuum, it does not predict a broad pattern of negative associations between standard diagnostic categories, but only between specific conditions or (more often) disorder subtypes. Moreover, the ubiquitous defense activation disorders (a category similar, but not identical, to the internalizing spectrum) overlap with both fast and slow spectrum conditions, while cognitive impairment and other nonspecific factors tend to create positive associations across spectra, partly masking the underlying negative associations.

As a result, the FSD model is fully compatible with the observed comorbidity structure of psychopathology. This can be demonstrated constructively by means of simulation: when the model is used to generate data based on standard diagnostic categories, and those data are analyzed with factor-analytic techniques, the results closely reproduce the structure found in empirical datasets—including two internalizing and externalizing factors as well as a general p factor (Del Giudice, 2016). Crucially, this happens even if the p factor as such is not part of the generating model: in the FSD, "p" emerges from the combination of three largely independent aspects of individual variation—fast life history strategies, heightened defense activation, and reduced cognitive ability (see Del Giudice & Haltigan, 2023). In this sense, the FSD model allows for an even deeper deconstruction (and reconstruction) of "p," one that goes beyond descriptive patterns of association—rich and informative as they may be—to embrace the challenge and power of functional explanation.

References

Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding "p." *Journal of Psychopathology and Clinical Science*, *135*(4), 461–494. https://doi.org/10.1037/abn0001042

Del Giudice, M. (2016). The life history model of psychopathology explains the structure of psychiatric disorders and the emergence of the p factor: A simulation study. *Clinical Psychological Science*, *4*(2), 299–311. https://doi.org/10.1177/2167702615583628

Del Giudice, M. (2018). *Evolutionary psychopathology: A unified approach*. Oxford University Press.

Del Giudice, M. (2020). Rethinking the fast-slow continuum of individual differences. *Evolution and Human Behavior*, *41*(6), 536–549. https://doi.org/10.1016/j.evolhumbehav.2020.05.004

Del Giudice, M. (2025). A turning point for the life history approach to individual differences. In S. Kanazawa (Ed.), *Genes, environments, and differential susceptibility: Current topics in evolutionary developmental psychology* (pp. 28–57). Cambridge University Press.

Del Giudice, M., & Haltigan, J. D. (2023). An integrative evolutionary framework for psychopathology. *Development and Psychopathology*, *35*(1), 1–11. https://doi.org/10.1017/S0954579421000870

Haeffel, G. J., Jeronimus, B. F., Fisher, A. J., Kaiser, B. N., Weaver, L. J., Vargas, I., Goodson, J. T., Soyster, P. D., & Lu, W. (2022). The

Hierarchical Taxonomy of Psychopathology (HiTOP) is not an improvement over the DSM. *Clinical Psychological Science*, *10*(2), 285–290. https://doi.org/10.1177/21677026211068873

Haeffel, G. J., Jeronimus, B. F., Kaiser, B. N., Weaver, L. J., Soyster, P. D., Fisher, A. J., Vargas, I., Goodson, J. T., & Lu, W. (2022). Folk classification and factor rotations: Whales, sharks, and the problems with the Hierarchical Taxonomy of Psychopathology (HiTOP). *Clinical Psychological Science*, *10*(2), 259–278. https://doi.org/10.1177/21677026211002500

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REPLY

What Does Psychopathology Research Have to Gain by Studying All Mental Disorders at the Same Time?

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The 16 constructive commentaries about our target article offer fresh ideas, new directions, and research questions. Here we amplify points raised about transdiagnostic approaches to etiology, treatment, and prevention; the role of cognitive ability in studying and treating mental disorders; the need to evaluate generational cohort changes in mental disorders; and the importance of assortative mating in life-course research and in treatment. We conclude by laying out a set of domains that would make sense for developing a p-informed measure of life-course vulnerability to psychopathology.

General Scientific Summary
In our target article, we made a case for why psychopathology research should avoid studying one mental disorder at a time. In responding to the thoughtful commentaries, here we try to show what psychopathology research has to gain by studying all mental disorders at the same time.

Keywords: structure of psychopathology, transdiagnostic approaches, developmental psychopathology

Discourse has become pugnacious in many spheres of life today, and debates in psychology—and among psychological scientists—likewise often turn quarrelsome. Against this background, we welcome the uniformly constructive commentaries about our article. As a set, the commentaries fuel an agenda for research. They are brimming with fresh ideas, new directions, and research questions. We amplify several of the questions raised by flipping the title of our target article to ask: *What does psychopathology research have to gain by studying all mental disorders at the same time?*

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Implications of a Transdiagnostic Approach for Etiology, Treatment, and Prevention

Specificity is one of the nine criteria, or "viewpoints," which epidemiologists have historically used to evaluate whether an association between a risk factor and an outcome is likely to be causal (Hill, 1965). The idea is that if a risk factor is associated with many dissimilar outcomes it is less likely to be causal, and more likely to be a product of confounding, than if it is associated with fewer outcomes. This criterion is problematic (as attested by historical debates about tobacco smoking, which is both associated with many outcomes and causes them; Parascandola, 2014). Nevertheless, psychopathologists continue to seek evidence of specificity (one-to-one relationships between causes and disorders), whether through research design (e.g., case-control studies) or statistical procedures (e.g., controlling for comorbidity). Even in those instances where transdiagnostic discoveries have been made, these came against the background of seeking disorder-specific discoveries. For example, a recent study identified five highly correlated genetic factors that account for the majority of variance across 14 different psychiatric disorders, suggesting that the same genetic factors may influence multiple different conditions (Grotzinger et al., 2026). This exemplar of cooperative science (Harden, 2026) demonstrates the value of looking across disorders rather than studying one disorder at a time. However, it bears noting that the author list of this article is made up of working groups of the Psychiatric Genomics Consortium, each of which was originally focused on a different disorder when the Psychiatric Genomics Consortium began in 2007: Schizophrenia Working Group, Bipolar, Eating, Depression, and so on. Would we have gotten to the 2026 genetic finding faster by studying multiple disorders at the same time? Looking forward, can new insights be achieved faster by studies that collect comprehensive mental health data on multiple dimensions of psychopathology rather than sampling one or a couple of disorders at a time?

Identifying transdiagnostic causal factors is an etiological goal, but it can also inform more effective mental-health treatments. Targeting vulnerability factors that cut across disorders (e.g., emotion dysregulation) could reduce both current and future mental-health problems, when balanced against components that are tailored to an individual's current presentation. Determining how to best achieve this balance represents an important aim of the next, "Third Wave" of psychological treatments (Dalgleish, 2026). Intervening on universal risks is also resource-efficient (Mansell, 2026), which is necessary given the gap between treatment need and availability and the siloing of mental health care (McGinty & Eisenberg, 2022).

Although the question of specificity has traditionally focused on causal factors, it is equally relevant to downstream effects: Different mental disorders often have the same consequences. Consider unintentional physical injuries (i.e., injuries for which there is no evidence of predetermined intent) and also injuries due to assaults. Whereas injury researchers have focused on specific disorders, such as substance use and attention-deficit/hyperactivity disorder [ADHD], we recently generated a comprehensive evidence base about the link between multiple different mental disorders and physical injuries (Richmond-Rakerd et al., 2026). Multiple mental disorders were all associated with an elevated likelihood of being injured. Of special interest, multiple mental disorders were consistently associated with trauma to the head and face, a finding that offers a window to identifying mechanisms by which multiple mental disorders in early life might shape brain health in later life. More generally, the findings suggest that injury psychoeducation could become a feature of transdiagnostic interventions.

Not only treatment but also prevention needs to become transdiagnostic. For this to happen, risk screening tools are needed that can capture transdiagnostic factors—a Framingham-style risk score for mental health (D'Agostino et al., 2013). Also needed are screening tools for overall psychopathology (p) that take a multiple mental-disorder approach (Neulinger et al., 2024). This message has found its way into clinical settings. For example, readers who have visited American primary-care and hospital clinics may have noticed that over the past decade the content of intake questionnaires has broadened considerably to inquire not only about depression but also about anxiety, substance use, concentration difficulties, and so forth. Along these lines, Tackett and Katz (2026) draw attention to the possibilities afforded by integrating personality assessments into models of psychopathology and possibly into risk assessment. As pointed out in the commentaries, tools that screen for overall mental illness or that capture p (Moore et al., 2019) are not a substitute for specific diagnoses, but they can inform prognosis and treatment planning (Pettersson, 2026; Pettersson et al., 2020).

Cognitive Ability

Our target article did not address cognitive abilities (Wilson et al., 2026), a significant omission given evidence that low IQ and mental disorders cluster in families (Weiser et al., 2023). There are at least three ways to incorporate information about cognitive ability into psychopathology and clinical science. First, in relation to nosology, information about cognitive dysfunction can be integrated into structural models of psychopathology. This kind of research is vital to better understand whether certain cognitive functions are more impaired in some disorders than in others and to identify cognitive functions that are impaired transdiagnostically (Ringwald et al., 2025).

Second, in relation to etiology, low cognitive ability has been proposed as a causal factor in the development of mental disorders (Barnett et al., 2006). Evidence from cognitive epidemiology (Deary & Batty, 2007) shows that low IQ is related to the risk of developing practically all mental disorders (Fries et al., 2025). For example, we recently examined the longitudinal associations between cognitive ability and mental disorders using military conscript test data from 18-year old Norwegian men (N = 272,351) and mental-disorder data from primary-care registers 20 years later (Nordmo et al., 2025). Lower cognitive ability was associated with a monotonically increasing risk of developing all the studied mental disorders (except bipolar disorder, a fascinating anomaly that has been observed in several studies [e.g., Gale et al., 2013; Koenen et al., 2009; Smith et al., 2015]). The associations were independent of educational attainment and held even when comparing the cognitive abilities of brothers raised in the same family, attesting that lower cognitive ability is not only associated with mental disorders because both arise from the same family-background circumstances. This aligns with evidence that cognitive difficulties characterize p. Individuals with higher levels of p fare less well on tests requiring attention, concentration, memory, as well as visual-perceptual processing speed and visual–motor

coordination (Caspi et al., 2014; Castellanos-Ryan et al., 2016; Martel et al., 2017). These cognitive difficulties are not simply a consequence of lifelong disorders; they are present already in early life, before the onset of most disorders (Caspi & Moffitt, 2018). Several factors, not mutually exclusive, could account for these transdiagnostic associations and warrant research. Lower IQ may be a marker of neuroanatomical and functional brain differences that affect executive function, attention, and processing speed and increase vulnerability to mental disorders. Individuals with lower IQs are also more likely to encounter stressful life events and are less equipped to cope with stressors, making them potentially more vulnerable after such events. Lower IQ may make activities of daily living more challenging and, therefore, stressful. Negotiating public transportation systems, comparison shopping, managing money, helping children with homework, and navigating the Internet are more demanding for individuals with lower IQs (Gottfredson, 1997). Finally, the association between IQ and mental disorders may be mediated by mental health knowledge, which facilitates early help-seeking, improving access to evidence-based care and promoting treatment compliance.

Third, in relation to treatment, even if they are not transdiagnostic causal factors, cognitive difficulties are complicating features that are important to consider in prevention and treatment planning. Interventions attempting to support and improve cognition (Harvey, 2025; Harvey et al., 2014) can be a useful complement to transdiagnostic interventions for emotional and behavioral disturbances.

Generational Cohort Effects

The absence of history in our target article is also notable (Keyes, 2026). After all, developmental trajectories are shaped by the intersection of individual lives, families, *and* historical contexts (Elder et al., 1988). Cohort effects refer to differences in health among people born during different historical periods (Rohrer, 2025). There is solid evidence that mental-health problems are on the rise among young people (Keyes & Platt, 2024), leading the American Psychological Association to declare a crisis. Most efforts to address the causes of increasing mental-health problems focus on specific conditions, such as autism, ADHD, or depression. But do proffered explanations for increasing rates of one disorder apply to other disorders? Are there explanations for increases that may be common across different disorders? Consider evidence about youth mental health, as documented in primary-care settings. For this reply, we studied the mental health of nine 1-year birth cohorts of children born between 2001 and 2009, following each cohort for 10 years from ages 5 to 15 years (Table 1 in the target article describes the mental-health conditions assessed in primary-care settings). Even over this short span, there were increases in the rates of both internalizing conditions (e.g., anxiety, depression) and externalizing conditions (e.g., ADHD) among children born more recently. The data suggest that these increases may not simply be an artifact of more young people visiting primary-care physicians as we did not observe this increase for somatic conditions or injuries (Figure 1A). An alternative approach to tackling the question of increasing rates of mental-health conditions one disorder at a time is to focus on three developmental parameters that covary within individuals. Together these three parameters signal a continuum of severity that differentiates between each person's mental-disorder life history: younger age-of-onset of disorder, longer life-course duration of disorder, and more diversity of disorders. These three parameters are core features of p (Caspi et al., 2020). We found that the age at first presenting to primary care for mental-health conditions has not become younger among children born more recently, but their mental-health conditions have become more persistent and more diverse (Figure 1B). This preliminary analysis is restricted to a brief historical period and a narrow developmental span, but it highlights how an emphasis on p rather than single disorders can reframe questions about causes and treatment.

Do explanations for the increasing rates of specific disorders (e.g., improved detection, greater awareness, changing diagnostic criteria, social media, eco-anxiety, and economic inequality) also explain the changing nature of mental-health trajectories among more recent birth cohorts? If these trajectories have really changed, we might also expect to see greater social, emotional, and occupational dysfunction (Atkinson et al., 2026). Indeed, such changing trajectories may align with increases in young people disengaged from education systems and the labor market (NEET: not in education, employment, or training) due to mental-health issues. Moreover, if the historical changes are not only disorder-specific changes but also changes in key developmental parameters that define p, it may be important to rethink service delivery. For example, if young people's mental-health conditions have become more persistent, greater emphasis would need to be placed in health care on moving young people from family-centered care to independent, patient-driven care as they grow older (Calabrese et al., 2022).

Assortative Mating

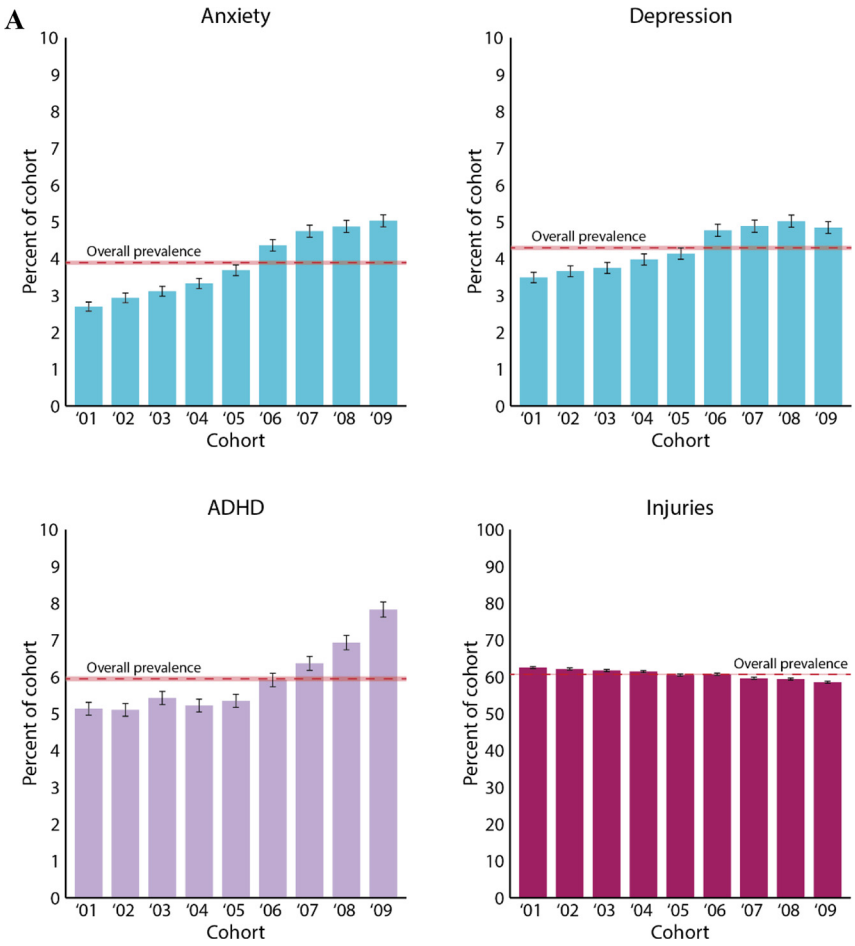
With notable exceptions (Border et al., 2022), studies of assortative mating typically focus on one disorder at a time. We documented that assortment was not specific to particular diagnostic categories, but we did not delve into transdiagnostic mechanisms (South, 2026).

It is unlikely that each mental disorder exists as a discrete etiological entity that only becomes correlated with other mental disorders after generations of assortative mating. Different disorders have too many shared risk factors for this to be the case. Indeed, assortment for mental disorders is often found to be indirect—based on related traits but not the observed diagnoses themselves (Torvik et al., 2024). This can lead to an entwining of all causal factors behind the traits that determine mate choice. In addition, gender differences in the expression of psychopathology and in partner preferences (Harper & Zietsch, 2025) suggest that partners may have different disorders even if they share similar risk factors. Moreover, diagnostic shifts within individuals imply that couples who have one combination of disorders at one point in time are at higher risk for all other combinations of disorders at later points in time. The partners in a couple remain the same, but the disorders they share shift. This also means that children rarely grow up with parents whose diagnostic statuses are stable throughout their entire childhood.

The implications of cross-disorder assortative mating for offspring are far-ranging, as assortative mating leads to a more unequal distribution of risk factors, passed on from both the mother and the father. This includes higher variance in p and greater covariance between different risk factors. Higher variance

Figure 1

Young People Born in More Recent Years Experience Distinct Mental-Health Trajectories



(*Figure continues*)

implies that there will be more offspring with very low or with very high values of p. An increase in variance most likely takes place when the degree of assortment is increasing, as has been shown for educational attainment and socioeconomic status (Sunde et al., 2024). However, it is uncertain how this weighs against reduced fertility among individuals who have mental disorders (Kravdal et al., 2025) and, presumably, higher p.

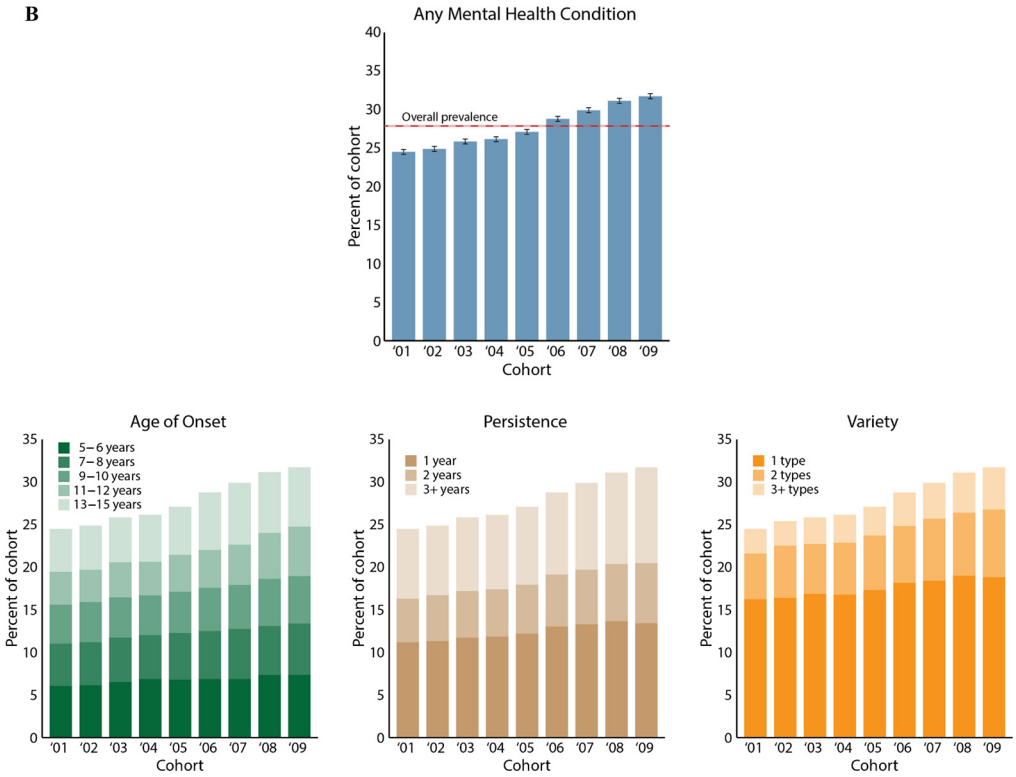
A particular case of correlated risk factors is covariance between the genetic and environmental components of multiple disorders (rGE). Such covariance means that the assumption of no gene-environment correlation, usually applied in genetic studies, does not hold. It also means that children who inherit a genetic risk for one disorder (e.g., depression) are more likely to live in environments created by parents with alcohol use disorder, OCD, and so forth. It is not straightforward to determine for which specific disorder these children are at risk. Instead, the risk constellation may lead to multiple, diverse, and unpredictable outcomes. Assortative mating therefore implies that p cuts not only across diagnostic boundaries but also across the nature-nurture divide.

Cross-disorder assortative mating and the familial clustering of different mental disorders also draw attention to the need to bridge youth and adult treatments, with the potential to disrupt the familial clustering of diverse types of psychopathology. For instance, there is encouraging evidence for transgenerational benefits of emotion regulation-focused interventions in parents (Zalewski et al., 2026), and both parents and their children may benefit from implementing similar skills to manage distressing symptoms (Ehrenreich-May & Milgram, 2026). However, many testable questions remain concerning which transdiagnostic approaches in youth and family therapy may work best and will be most acceptable to providers, as well as about potential limits to these approaches when specialized care may be needed (Ehrenreich-May & Milgram, 2026).

Should Our Article Have Provoked More Pushback?

Given that the idea of p has raised hackles (see Plutynski, 2026; for a philosophical perspective on the debate), it may surprise that the commentaries were not more critical. Why? One possibility relates to the division between the two audiences of this journal: psychopathologists and clinical scientists. psychopathologists, who focus on the measurement and causes of symptoms of mental disorder, have been debating whether p is a substantive construct, an index of overall mental illness, or a measurement or statistical artifact. In contrast, clinical scientists, who seek to develop effective

Figure 1 (*continued*)



Note. We observed the mental health of nine 1-year birth cohorts of children born between 2001 and 2009, following them for 10 years from ages 5 to 15 years. The sample sizes in each cohort range from 52,734 to 58,772. Panel A shows that both internalizing (e.g., anxiety, depression) and externalizing (e.g., ADHD) conditions have increased among youth. Injuries, shown for comparison, have not increased. Panel B shows that the age at first presenting to primary care for mental-health conditions has not become younger among children born more recently; if anything it has increased slightly (e.g., in the earliest cohort the average age was 9.23 years; in the most recent cohort the average age was 9.42 years). But their mental-health conditions have become more persistent, $F(8, 497{,}646) = 111.99$, $p < .001$, and more diverse, $F(8, 497{,}646) = 251.38$, $p < .001$. See the online article for the color version of this figure.

treatments for mental disorders, have left the skepticism behind. They seem to recognize p in their patients and their families.

Our article sought to document the three reasons that clinical scientists see p during their work (cross-disorder assortative mating, transdiagnostic intergenerational transmission, shifting disorders over the life course). We aimed to articulate what this means for how psychopathologists carry out their research, but our article did not take a strong stance about what p means.

Take athleticism as an example. Most of us would agree that there is such a thing as athleticism. But that does not mean there is a unitary mechanism that explains athleticism. Rather, it is made up of multiple different causes: jumping ability, running speed, oxygen capacity, fine motor control, gross motor control, response time, and balance. Different combinations of these "causes" lead some people to be better at some specific sports (basketball, track and field, gymnastics). And because many of these causes are correlated, people who are excellent in some sports are generally better than other people in all sports. But equally, just because athleticism is made up of different (albeit correlated) causes does not mean there is no such thing as athleticism. p does not explain co-occurring mental disorders any more than athleticism explains athletic ability. But it does suggest what might be common across many mental disorders, just like athleticism focuses our attention on what makes for athletic ability. It can also point to interventions with broad mental-health effects, just like improved exercise, rather than any specific physical activity, brings about widespread health benefits.

Raballo et al. (2026) summarize our thesis better than we have: "The p-factor, as statistically identified, is not a causal entity but rather the observable trace of a deeper vulnerability architecture, a developmental and social process that unfolds interactively across time and generations" (p. 536). By definition, p is transdiagnostic. A unified measurement tool is needed that can assess an individual's life-course vulnerability to psychopathology (De Los Reyes, 2026). Such a tool would draw attention to what all disorders share and also to what all people share, to a degree. Its elements would likely include at least these components: a *diffuse unpleasant affective state*, often termed neuroticism or negative emotionality; *poor impulse control* over thoughts (e.g., impulsive overgeneralization from negative events) and emotions (e.g., impulsive speech and action in response to experienced emotions); *thought distortion* characterized by reality-distorted and reality-distorting cognitions; *interpersonal impairments*, which are nonspecifically evident across many disorders; and *low cognitive ability* characterized by difficulties with

attention, concentration, memory, processing speed, visual-perceptual processing, and visual–motor coordination. These are not competing explanations or accounts but reinforcing dimensions of psychological dys/function. Also, p is a life-course phenomenon and an intergenerational phenomenon. As such, it draws attention away from efforts to predict which symptoms will emerge and for whom. Instead, it draws attention toward efforts to study symptoms as maladaptive deviations from typical developmental trajectories (Moriarity & Hanson, 2026). In research and treatment, such a functional perspective calls for adopting a developmental framework that focuses on how individuals contend with biosocial imperatives; changing age-graded roles; and unexpected, nonnormative events (Del Guidice, 2026; Hinshaw, 2026). This perspective is also most compatible with transdiagnostic staging models (Buchweitz et al., 2026) that are focused on the evolving nature of mental illness rather than cross-sectional diagnostics.

References

Atkinson, E. A., Oltmanns, J. R., & Smith, G. T. (2026). The value of focusing on psychosocial dysfunction rather than categorical disorders: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 515–517. https://doi.org/10.1037/abn0001088

Barnett, J. H., Salmond, C. H., Jones, P. B., & Sahakian, B. J. (2006). Cognitive reserve in neuropsychiatry. *Psychological Medicine*, *36*(8), 1053–1064. https://doi.org/10.1017/S0033291706007501

Border, R., Athanasiadis, G., Buil, A., Schork, A. J., Cai, N., Young, A. I., Werge, T., Flint, J., Kendler, K. S., Sankararaman, S., Dahl, A. W., & Zaitlen, N. A. (2022). Cross-trait assortative mating is widespread and inflates genetic correlation estimates. *Science*, *378*(6621), 754–761. https://doi.org/10.1126/science.abo2059

Buchweitz, C., Viduani, A., Herrman, H., Kohrt, B. A., McGorry, P., Salum, G., Sartor, C., Saxena, S., & Kieling, C. (2026). Transdiagnostic stage-based monitoring of public mental health. *Nature Reviews Psychology*, *5*(2), 120–135. https://doi.org/10.1038/s44159-025-00527-w

Calabrese, S., Lee, S., Mollica, M. A., Wiener, L., Mendley, S., Adams, L., & Blachman-Demner, D. (2022). Navigating pediatric to adult healthcare transition: A national institutes of health workshop. *The Journal of Pediatrics*, *244*, 234–240.e1. https://doi.org/10.1016/j.jpeds.2022.01.052

Caspi, A., & Moffitt, T. E. (2018). All for one and one for all: Mental disorders in one dimension. *The American Journal of Psychiatry*, *175*(9), 831–844. https://doi.org/10.1176/appi.ajp.2018.17121383

Caspi, A., Houts, R. M., Ambler, A., Danese, A., Elliott, M. L., Hariri, A., Harrington, H., Hogan, S., Poulton, R., Ramrakha, S., Rasmussen, L. J. H., Reuben, A., Richmond-Rakerd, L., Sugden, K., Wertz, J., Williams, B. S., & Moffitt, T. E. (2020). Longitudinal assessment of mental health disorders and comorbidities across 4 decades among participants in the Dunedin birth cohort study. *JAMA Network Open*, *3*(4), Article e203221. https://doi.org/10.1001/jamanetworkopen.2020.3221

Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., Meier, M. H., Ramrakha, S., Shalev, I., Poulton, R., & Moffitt, T. E. (2014). The p factor: One general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science*, *2*(2), 119–137. https://doi.org/10.1177/2167702613497473

Castellanos-Ryan, N., Brière, F. N., O'Leary-Barrett, M., Banaschewski, T., Bokde, A., Bromberg, U., Büchel, C., Flor, H., Frouin, V., Gallinat, J., Garavan, H., Martinot, J.-L., Nees, F., Paus, T., Pausova, Z., Rietschel, M., Smolka, M. N., Robbins, T. W., Whelan, R., … The IMAGEN Consortium. (2016). The structure of psychopathology in adolescence and its common personality and cognitive correlates. *Journal of Abnormal Psychology*, *125*(8), 1039–1052. https://doi.org/10.1037/abn0000193

D'Agostino, R. B., Sr, Pencina, M. J., Massaro, J. M., & Coady, S. (2013). Cardiovascular disease risk assessment: Insights from Framingham. *Global Heart*, *8*(1), 11–23. https://doi.org/10.1016/j.gheart.2013.01.001

Dalgleish, T. (2026). Toward a third wave of transdiagnostic psychological therapies: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 531–533. https://doi.org/10.1037/abn0001107

De Los Reyes, A. (2026). Toward integrative, multisource, and multimodal approaches to assessing psychopathology intergenerationally and across the lifespan: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 495–497. https://doi.org/10.1037/abn0001084

Deary, I. J., & Batty, G. D. (2007). Cognitive epidemiology. *Journal of Epidemiology and Community Health*, *61*(5), 378–384. https://doi.org/10.1136/jech.2005.039206

Del Guidice, M. (2026). Understanding and transcending "p" requires a functional model of psychopathology: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 507–509. https://doi.org/10.1037/abn0001080

Ehrenreich-May, J., & Milgram, L. (2026). The promise of transdiagnostic prevention and treatment for youth and caregiver psychopathology: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 510–512. https://doi.org/10.1037/abn0001085

Elder, G. H., Jr., Caspi, A., & Burton, L. (1988). Life transitions in developmental perspective: Sociological and historical insights on adolescence. In M. Gunnar & W. A. Collins (Eds.), *Minnesota symposia on child psychology* (Vol. 21). Erlbaum.

Fries, J., Oberleiter, S., Bodensteiner, F. A., Fries, N., & Pietschnig, J. (2025). Multilevel multiverse meta-analysis indicates lower IQ as a risk factor for physical and mental illness. *Communications Psychology*, *3*(1), Article 74. https://doi.org/10.1038/s44271-025-00245-2

Gale, C. R., Batty, G. D., McIntosh, A. M., Porteous, D. J., Deary, I. J., & Rasmussen, F. (2013). Is bipolar disorder more common in highly intelligent people? A cohort study of a million men. *Molecular Psychiatry*, *18*(2), 190–194. https://doi.org/10.1038/mp.2012.26

Gottfredson, L. S. (1997). Why g matters: The complexity of everyday life. *Intelligence*, *24*(1), 79–132. https://doi.org/10.1016/S0160-2896(97)90014-3

Grotzinger, A. D., Werme, J., Peyrot, W. J., Frei, O., de Leeuw, C., Bicks, L. K., Guo, Q., Margolis, M. P., Coombes, B. J., Batzler, A., Pazdernik, V., Biernacka, J. M., Andreassen, O. A., Anttila, V., Børglum, A. D., Breen, G., Cai, N., Demontis, D., Edenberg, H. J., … Substance Use Disorders Working Group of the Psychiatric Genomics Consortium. (2026). Mapping the genetic landscape across 14 psychiatric disorders. *Nature*, *649*(8096), 406–415. https://doi.org/10.1038/s41586-025-09820-3

Harden, K. P. (2026). In praise of scientific open relationships: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 505–506. https://doi.org/10.1037/abn0001079

Harper, K. T., & Zietsch, B. P. (2025). Assortative mating is a natural consequence of heritable variation in preferences and preferred traits. *Psychological Science*, *36*(10), 771–779. https://doi.org/10.1177/09567976251365900

Harvey, A. G., Lee, J., Williams, J., Hollon, S. D., Walker, M. P., Thompson, M. A., & Smith, R. (2014). Improving outcome of psychosocial treatments by enhancing memory and learning. *Perspectives on Psychological Science*, *9*(2), 161–179. https://doi.org/10.1177/1745691614521781

Harvey, A. G. (2025). Maximizing benefits from evidence-based psychological treatments: Memory support and habit formation as key strategies. *Behaviour Research and Therapy*, *191*, Article 104767. https://doi.org/10.1016/j.brat.2025.104767

Hill, A. B. (1965). The Environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine*, *58*(5), 295–300. https://doi.org/10.1177/003591576505800503

Hinshaw, S. P. (2026). Multiple disorders across individual development: The role of heterotypic continuity—Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 502–504. https://doi.org/10.1037/abn0001090

Keyes, K. M. (2026). On the cross-generational expression of psychiatric disorders: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 500–501. https://doi.org/10.1037/abn0001087

Keyes, K. M., & Platt, J. M. (2024). Annual research review: Sex, gender, and internalizing conditions among adolescents in the 21st century - Trends, causes, consequences. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *65*(4), 384–407. https://doi.org/10.1111/jcpp.13864

Koenen, K. C., Moffitt, T. E., Roberts, A. L., Martin, L. T., Kubzansky, L., Harrington, H. L., Poulton, R., & Caspi, A. (2009). Childhood IQ and adult mental disorders: A test of the cognitive reserve hypothesis. *The American Journal of Psychiatry*, *166*(1), 50–57. https://doi.org/10.1176/appi.ajp.2008.08030343

Kravdal, Ø., Flatø, M., & Torvik, F. A. (2025). Fertility among Norwegian women and men with mental disorders. *European Journal of Population*, *41*(1), Article 17. https://doi.org/10.1007/s10680-025-09739-5

Mansell, W. (2026). Shared transdiagnostic risks are universal, dynamic, and connected: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 498–499. https://doi.org/10.1037/abn0001076

Martel, M. M., Pan, P. M., Hoffmann, M. S., Gadelha, A., do Rosário, M. C., Mari, J. J., Manfro, G. G., Miguel, E. C., Paus, T., Bressan, R. A., Rohde, L. A., & Salum, G. A. (2017). A general psychopathology factor (P factor) in children: Structural model analysis and external validation through familial risk and child global executive function. *Journal of Abnormal Psychology*, *126*(1), 137–148. https://doi.org/10.1037/abn0000205

McGinty, E. E., & Eisenberg, M. D. (2022). Mental health treatment gap: The implementation problem as a research problem. *JAMA Psychiatry*, *79*(8), 746–747. https://doi.org/10.1001/jamapsychiatry.2022.1468

Moore, T., Calkins, M. E., Satterthwaite, T. D., Roalf, D. R., Rosen, A. F. G., Gur, R. C., & Gur, R. (2019). Development of a computerized adaptive screening tool for overall psychopathology ("p"). *Journal of Psychiatric Research*, *116*, 26–33. https://doi.org/10.1016/j.jpsychires.2019.05.028

Moriarity, D. P., & Hanson, J. L. (2026). Shuffling through hierarchies in developmental psychopathology: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 528–530. https://doi.org/10.1037/abn0001096

Neulinger, B., Ebert, C., Lochbühler, K., Bergmann, A., Gensichen, J., & Lukaschek, K. (2024). Screening tools assessing mental illness in primary care: A systematic review. *The European Journal of General Practice*, *30*(1), Article 2418299. https://doi.org/10.1080/13814788.2024.2418299

Nordmo, M., Sunde, H. F., Kleppestø, T. H., Nordmo, M., Caspi, A., Moffitt, T. E., & Torvik, F. A. (2025). Cognitive abilities and educational attainment as antecedents of mental disorders: A total population study of males. *Psychological Science*, *36*(7), 499–513. https://doi.org/10.1177/09567976251347221

Parascandola, M. (2014). Commentary: Smoking, birthweight and mortality: Jacob Yerushalmy on self-selection and the pitfalls of causal inference. *International Journal of Epidemiology*, *43*(5), 1373–1377. https://doi.org/10.1093/ije/dyu163

Pettersson, E. (2026). The p-factor and associations across partners, time, and generations: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 513–514. https://doi.org/10.1037/abn0001086

Pettersson, E., Larsson, H., D'Onofrio, B. M., Bölte, S., & Lichtenstein, P. (2020). The general factor of psychopathology: A comparison with the general factor of intelligence with respect to magnitude and predictive validity. *World Psychiatry*, *19*(2), 206–213. https://doi.org/10.1002/wps.20763

Plutynski, A. (2026). The meaning of a measure: P as a general measure of psychopathology. *Philosophical Psychology*, 1–33. https://doi.org/10.1080/09515089.2025.2611430

Raballo, A., Poletti, M., & Preti, A. (2026). Rethinking the p-factor: A model of cumulative risk aggregation across development—Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 534–536. https://doi.org/10.1037/abn0001112

Richmond-Rakerd, L. S., Milne, B. J., Houts, R. M., Davie, G., D'Souza, S., Goldman-Mellor, S., Khalifeh, L., Caspi, A., Moffitt, T. E., & Torvik, F. A. (2026). Mental health conditions are associated with increased risk of subsequent self-harm, assault and unintentional injuries in two nations. *Nature Mental Health*, *4*(1), 102–111. https://doi.org/10.1038/s44220-025-00553-w

Ringwald, W. R., Abramovitch, A., Agelink van Rentergem, J. A., & Kotov, R. (2025). Do cognitive functions belong in the hierarchical taxonomy of psychopathology model? A meta-analysis. *Perspectives on Psychological Science*, *20*(6), 1049–1064. https://doi.org/10.1177/17456916251347926

Rohrer, J. M. (2025). Thinking clearly about age, period, and cohort effects. *Advances in Methods and Practices in Psychological Science*, *8*(2). https://doi.org/10.1177/25152459251342750

Smith, D. J., Anderson, J., Zammit, S., Meyer, T. D., Pell, J. P., & Mackay, D. (2015). Childhood IQ and risk of bipolar disorder in adulthood: Prospective birth cohort study. *BJPsych Open*, *1*(1), 74–80. https://doi.org/10.1192/bjpo.bp.115.000455

South, S. C. (2026). The relationship's the thing: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 518–519. https://doi.org/10.1037/abn0001089

Sunde, H. F., Eftedal, N. H., Cheesman, R., Corfield, E. C., Kleppesto, T. H., Seierstad, A. C., Ystrom, E., Eilertsen, E. M., & Torvik, F. A. (2024). Genetic similarity between relatives provides evidence on the presence and history of assortative mating. *Nature Communications*, *15*(1), Article 2641. https://doi.org/10.1038/s41467-024-46939-9

Tackett, J. L., & Katz, B. A. (2026). P is for personality: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 526–527. https://doi.org/10.1037/abn0001092

Torvik, F. A., Sunde, H. F., Cheesman, R., Eftedal, N. H., Keller, M. C., Ystrom, E., & Eilertsen, E. M. (2024). Non-random mating patterns within and across education and mental and somatic health. *Nature Communications*, *15*(1), Article 10505. https://doi.org/10.1038/s41467-024-54966-9

Weiser, M., Frenkel, O., Fenchel, D., Tzur, D., Sandin, S., Janecka, M., Levi, L., Davidson, M., Laor, L., Fruchter, E., & Reichenberg, A. (2023). Familial clustering of psychiatric disorders and low IQ. *Psychological Medicine*, *53*(7), 2878–2884. https://doi.org/10.1017/S0033291721004852

Wilson, S., Ringwald, W., & Michelini, G. (2026). Transdiagnostic psychopathology research requires comprehensive, lifespan coverage of psychological dysfunction *and* function and ability: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 523–525. https://doi.org/10.1037/abn0001094

Zalewski, M., Crowell, S. E., & Stepp, S. D. (2026). Transdiagnostic formulations can inform intervention and academic training program: Commentary on Caspi et al. (2026). *Journal of Psychopathology and Clinical Science*, *135*(4), 520–522. https://doi.org/10.1037/abn0001091

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